PREVALENCE OF "CONGENITAL LUMBAR SPINAL STENOSIS" IN PATIENTS WITH CHRONIC LOW BACK PAIN IN MOMBASA

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ABSTRACT

Background: Chronic low back pain is one of the commonest maladies of man. There are multiple causes of chronic low back pain that will include degenerative, inflammatory and mechanical causes. Developmental lumbar spinal stenosis is known to cause symptoms of axial back pain with or without leg pain in the young adult. These symptoms become severer when patients with developmental stenosis acquire degenerative changes as the severity of theca sac and foramina compression increases. We hypothesized that developmental lumbar spinal stenosis is a major predisposing factor for chronic low back pain in adult population.

Objective: The purpose of this study was to evaluate the prevalence of developmental lumbar spinal stenosis in a group of individuals suffering from chronic low back pain. This prevalence was compared with another group of asymptomatic individuals (without low back pain). Both Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) images were used for this analysis.

Design: This was a prospective, case-control, radiographic study.

Subjects: Radiological materials from 118 individuals undergoing MRI scans for chronic low back pain with or without leg pain were analyzed to obtain the AP diameters of the lumbar vertebral body and vertebral canals at L4 and L5 levels. Ninety six patients were enrolled in this study. Abdominal CT scans of 96 patients without back pain were obtained from radiological archives for use as controls.

Methods: Using simple statistical methods the association between developmental lumbar spinal stenosis and chronic low back pain was examined.

Results: In the study group, 26% of the participants had canal stenosis (AP diameter <12mm) at the distal lumbar canal compared to 8% in the control group; 38% had moderate sized canal (12-14mm) in the study group (31% in control) while only 36% had a normal canal (66% in control group). The differences were found to be statistically significant (95% CI 0.4-1 P=0.0000023). In this study 64% of the patients had a canal diameter below the mean. The presence of a narrow canal is prevalent in these patients with chronic low back pain (Odds ratio 0.3). There was weak correlation between size of the body and size of the canal (Pearson's r = 0.4).

Conclusion: There is significant association between developmental distal lumbar canal stenosis and chronic low back pain in adults.

INTRODUCTION

There are a multitude of causes for Low Back Pain (LBP). In a study evaluating the pathophysiology of back pain presenting to a primary care physician, 4% of patients had a compression fracture, 3% had spondylolisthesis, 0.7% had a tumour, 0.3% had ankylosing spondylitis, and 0.01% had an infection. The overwhelming cause of back pain remained nonspecific (1, 2). It is in this nonspecific group that we believe we find congenital lumbar spinal stenosis as a major participant.

Spinal stenosis whether congenital or acquired is defined as a narrowing of the spinal canal (vertebral canal) by either the bony cage or a combination of bone and soft tissues, which causes mechanical compression of the theca sac and or spinal nerve roots. However, congenital (or developmental) Lumbar Stenosis (LSS) and the acquired (or degenerative) type are distinct from one another and although this distinctness is generally acknowledged (3), degenerative changes will make a hitherto quiescent congenital type symptomatic. The compression of the nerve roots may remain asymptomatic in childhood, but eventually become symptomatic in adulthood. These symptoms include: muscle weakness, reflex alterations, gait disturbances, bowel or bladder dysfunction, motor and sensory changes, radicular pain or atypical leg pain, and neurogenic claudication.

Very little is known about the epidemiology of congenital stenosis in the general population despite the fact that lumbar spinal stenosis is one of the most commonly diagnosed and treated conditions affecting the spine. The pathoaetiology, predisposition and the clinical syndrome seen in adults with narrow spinal canals as opposed to those with acquired or degenerative spines is unknown. There is no universally accepted diagnostic criterion for spinal stenosis (4).

This is a major difficulty in performing an epidemiologic analysis. Computerized imaging (particularly MRI and CT scanning) are most frequently utilized modalities for diagnosis in clinical practice. Recognizing these limitations, we developed a criterion for classifying congenital stenosis according to the canal diameters measured in scaled CT or MRI scans. Apart from direct measurement of canal diameter, these imaging modalities also reveal abnormal developmental changes in the vertebrae and of particular interest are spina bifidas and trefoilness. These three anomalies are clinically important when affected spines are compromised further by other pathologies. It is not surprising therefore, that symptomatic disc protrusion is more common in patients with trefoil shaped vertebral canals, where space is at a premium, than in the general population, and it is less common in patients with spina bifida occulta and with isthmic spondylolisthesis where the canal is more spacious. Acquired changes such as disc degeneration, hypertrophied ligamentum flavum, listhesis and spondylolysis, subarticular and or foramina narrowing in an already stenotic canal can only worsen the symptoms.

MATERIALS AND METHODS

Study design: A prospective, case-control, radiographic study.

Patient sample: All study participants were voluntary patients of the author between July 2010 and December 2013. All underwent a thorough clinical assessment including filling in the validated and widely used modified Nordic Low Back Questionnaire (5). The questionnaire defines significant LBP as "low back pain on most days of at least one month in the last 12 months". Recorded neurological symptoms included saddle anaesthesia, bowel or bladder disturbance, pain in the buttocks or thighs or below the knee, numbness or tingling in the leg or foot, or weakness in the leg or foot. Lumbosacral plain radiographs and Magnetic Resonance Imaging (MRI) scans (axial and sagittal) were then done on all the patients with a clinical diagnosis of LBP. All the MRI scans were done using a GE 1.5 Teslar Scanner (2008). Out of 222 patients 96 cases were non-randomly selected to match (for age, sex and ethnicity) with controls. Soft copies of 154 abdominal CT scans done between the year 2012 and December 2013 were retrieved. All the CT scans were done using a Siemens Somatom Perspective 128 CT Scanner (2012). The participants' clinical records were also retrieved to confirm the diagnosis. Out of 154 records, we were able to get 96 participants with readable scans and who had no history of back pain. These scans were used to control the study. Patients with severe degenerative changes, osteoporosis and deformity were excluded. Excluded too were 3 cases of spine infection and 2 cases of metastatic disease.

Outcome measures: MRIs and CT scans were assessed by the author. Both MRI and CT scans were scaled at

source to allow measurement of the desired parameters. Measurements were done using appropriate computer software (Painter Image editor). Bone windows were used for measurements. The antero-posterior diameter of the spinal canal was measured at the interpedicular level. This level is considered more precise than the midsagittal due to avoidance of inaccurate measurements resulting from scoliosis or improper patient positioning (6). Similarly, Antero-Posterior (AP) measurements of the vertebral body diameter of L4 and L5 vertebral bodies were done.

RESULTS

Analysis: The results of measurement on the images of the 192 individuals were tabulated in worksheets. The results were then grouped and graded utilizing a fourtier grading scales as shown in Tables 1 and 2.

 Table 1

 Showing the four- tier grading for the spinal canal size

| <10 | Measurement | Description |
|-----------------------|-------------|-------------------|
| 12.1-14 Mild stenosis | <10 | Severe stenosis |
| | 10.1 - 12 | Moderate stenosis |
| Normal | 12.1-14 | Mild stenosis |
| -14 INOIIIIai | >14 | Normal |

Table 2

Showing the four- tier grading for the vertebral body

| size | | | |
|--------------|----------------|--|--|
| VB size (mm) | Description | | |
| <25 | Underdeveloped | | |
| 25 - 30 | Small | | |
| 31-35 | Average | | |
| >36 | Normal | | |

Statistical analysis: All statistical analyses were performed using IBM SPSS Statistics 19 and other computer based programs.

The study sample included 96 study participants, 37 (39%) males and 59 (61%) females. The mean age was 39.3 ± 8.2 (age range: 18–50). In the control group there were 41(43%) males and 55 (57%) females; the mean age in the group was 48.7 ± 17.2 . The comparison tests (F.TEST) showed no age or sex difference between the study and the control groups (p=2.7999). In the study group all participants reported experiencing LBP on most days of at least one month in the last 12 months. Most patients presented with multiple symptoms that are characteristic of low back pain syndrome. The distribution of symptoms is as shown in Table 3.

| Showing the frequency | of sympton | ms |
|--------------------------------|------------|-----|
| Symptom | No. | (%) |
| Back pain and stiffness | 73 | 76 |
| Pain in lower leg (below knee) | 56 | 58 |
| Numbness in the leg or foot | 43 | 45 |
| Pain in a buttocks or thighs | 13 | 14 |
| Bowel and bladder disturbance | 10 | 10 |
| Weakness in the leg or foot | 9 | 9 |
| Claudication | 7 | 7 |

 Table 3

 Showing the frequency of symptoms

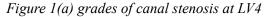
The AP diameter of the canal was significantly smaller in the low back pain patients at both lumbar levels than in the control group (L4: 14.9 mm (0.7) vs. 15.3 mm (0.7), p = 0.4514; and at L5: 13.0mm (0.7) vs. 14.7 mm (0.8), p = 0.0071) (Table 4).

Table 4Spinal canal diameter (a) comparing the means atLV4 (b) in LV5

| | Confidence | | | |
|-----------------|------------|----------|--------|--|
| Canal size (mm) | Mean | interval | T test | |
| Canal size LV4 | | | | |
| Study | 14.9 | 0.7 | | |
| Control | 15.3 | 0.7 | 0.4514 | |
| Canal size LV5 | | | | |
| Study | 13.0 | 0.7 | | |
| Control | 14.7 | 0.8 | 0.0071 | |

The differences at L5 level were statically significant. The distribution of various grades of stenosis in the study and control samples was done separately for LV4 and LV5. The results are shown in Figures 1(a) and 1(b).

Figure 1 Distribution in various grades of stenosis



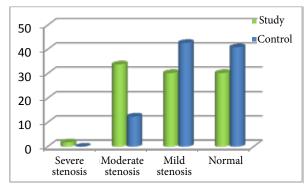
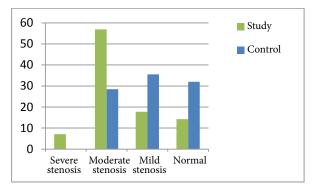
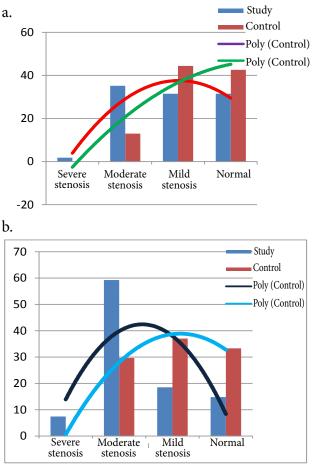


Figure 1(b) grades of canal stenosis at LV5



In the study group, absolute stenosis (AP diameter < 10 mm) was observed in 4 (7%) at LV5 level and in 2 (2%) at LV54 level; whereas there were no absolute stenosis in the control group. With a 12 mm AP diameter cut-off between normal and stenotic canals, then 28 (26%) of the study group participants had distal lumbar stenosis compared to 9 (8%) in the control group. Therefore, 26% of the LBP had a distal lumbar canal of less than 12mm in comparison to the control group where only 8% of the control group had a canal less than 12 mm (Figure 2(a) and (b).

Figure 2 Grades of canal stenosis (a) frequency of distribution LV4 which is almost binomial, while (b) distribution at LV5 shows a shift to the left



Our study shows no difference in prevalence of radiographic LSS between men and women (Male 13.8 mm (3.2), Female 14.1(2.2) P = 0.66369).

| Table 5 |
|--|
| The odds ratio for CLSS between the two groups |

| Summary of canal diameter at 14/15 | | | | | |
|------------------------------------|---------------|----------------|-------|---------|-----------|
| Canal size | Control group | Study group | Total | CI | P value |
| <12 mm 8 21 29 1.11007 | | | | | |
| >12 mm | 88 | 75 | 163 | 0.35616 | |
| Mean | 96 | 96 | 192 | | 0.0000023 |
| Odds ratio (or) $= 0.3$ | | | | | |

Similarly the vertebral body length was markedly shorter in the LBP group at both lumbar levels, although the differences were not statically significant (LV4: 30.3 mm (0.9) vs. 31.2 mm (1.3), p = 0.2713); and at LV5: 31.2 mm (1.0) vs. 32.1 mm (2.2), p = 0.2551).

Table 6Vertebral body diameter (a) comparing the means atLV4 (b) in LV5

| VB size (mm) | Mean | Confidence I | T test |
|-------------------------|------|--------------|--------|
| Vertebral body size LV4 | | | |
| Study | 30.3 | 0.9 | |
| Control | 31.2 | 1.3 | 0.2713 |
| Vertebral body size LV5 | | | |
| Study | 31.2 | 1.0 | |
| Control | 32.1 | 2.2 | 0.2551 |

There was a weak correlation between AP diameter of the vertebral body and of the spinal canal (Pearson product moment correlation coefficient: r = 0.4).

DISCUSSION

Low back pain is one of the most common disorders of mankind. Although back pain is ubiquitous, more than 70% of people in developed countries experience low back pain at some time in their lives (7). Every year, one third to one half of adults suffers low back pain and 5% of people present to their medical provider with a new episode. Low back pain is most common in patients between the ages of 35 and 55 years (7). Low back pain is also a major cause of pain and disability and a common indication for spine surgery. In the developing world, where spine surgery is not readily available, degenerative spine disorders are a major cause of deformity such as stooping, humping, general shortening and premature aging. The contribution of congenital spinal stenosis to low back pain has not been quantified. The disease process usually begins with degeneration of the intervertebral disks and facet joints, resulting in narrowing of the spinal canal and neural foramina. Associated factors may include a developmentally narrow spinal canal and degenerative spinal instability. Hilibrand and Rand (8) reported in a short-term follow-up data of surgically decompressed patients showed superiority of operative management over non-operative treatment, perhaps, suggesting that morbidity is more related to narrowness than inflammation or instability. They reported surgical success rates as high as 85%.

We conducted a study intended to relate the prevalence of congenital lumbar spine stenosis among low back pain patients. We used MRI scans to measure the AP diameter of the lumbar canal at LV4 and LV5 levels in patients with chronic low back pain. Bone windows were used for both measurements. The Antero-Posterior diameter (AP) of the spinal canal was measured at the mid-vertebral body level. This level is considered more precise than the mid-sagittal view due to avoidance of inaccurate measurements resulting from scoliosis or improper patient positioning (6). Similarly, measurements of the AP vertebral body diameter from the axial MRIs scans were done on L4 and L5 vertebral body only. For the control group, similar measurements were done on CT scans using appropriate computer software (Painter Image editor). We developed a criteria where <10mm was considered severe stenosis (absolute stenosis), 10.1-12mm moderate stenosis, 12.1-14 mm mild stenosis and >14 mm was considered normal. These parameters are on the lower side of published data. Studies done among the Arabs in Egypt and Lebanon show a mean canal depth of 15.6 mm at L5 (9,10). Eisenstein (11) in a multiethnic study in South Africa found a mean midsagittal diameter at L5 of 15 mm. In this study, we concluded that LSS is more prevalent at LV5 than LV4 and that individuals with AP canal diameters ≤ 12 mm, particularly at LV5 have a statistically significant association between LSS and occurrence of LBP (odds ratio (OR=0.3 (95% CI: 0.4-0.7)). Kern Singh et al (12) used a cross-sectional area of the canal in 20 symptomatic congenitally stenotic individuals and ageand sex-matched with 20 asymptomatic, nonstenotic individuals. They showed that the cross-sectional area of the canal was significantly smaller in the congenitally stenotic patients at all lumbar levels measured (LV2-LV5). However, little is known regarding the epidemiology of congenital spinal stenosis in the general population. Verbiest (13) measured the midsagittal diameter of the lumbar canal at operation and proposed two major types of stenosis: absolute stenosis, with diameter 10 mm or less; and relative stenosis with diameters ranging from 10 to 12 mm. In a CT study, the same author suggested that mid-sagittal lumbar canal

diameters less than 10 mm represent absolute stenosis and diameters less than 13 mm represent relative stenosis (13). Ulrich and colleagues (6) suggested that the antero-posterior diameter of the spinal canal (measured on axial plain CT) of less than 11.5 mm is small. In another CT study, Lee and colleagues (14) reported that the sagittal diameter of the lumbar spinal canal is never smaller than 10 mm in a normal spine. Haig *et al.* (15) demonstrated that antero-posterior measurements of the spinal canal (using 11.95 mm as a threshold) can distinguish between patients with clinical spinal stenosis and asymptomatic individuals.

The association between LSS and LBP has been studied in the past but not fully established. De Villiers and Booysen (16) in a report of 850 myelograms found a 6% prevalence of lumbar spinal stenosis but did not separate the congenital from the acquired forms of this condition. Fanuele et al. (17) reported a prevalence of 13.1% among 17,744 patients. This was a large study utilizing a multicenter clinical database without providing criteria for diagnosis of lumbar canal stenosis. In a multicentre study confounders (such as genetic and environmental factors) are not eliminated. Our study selected patients from one locality who were living under similar environmental circumstances. Our study found 8% of control patients were asymptomatic despite a narrow canal. We should have analysed this group further, particularly for age but the numbers were small. However, there have been multiple studies reporting the occurrence of congenital spinal stenosis in asymptomatic individuals. Haig and colleagues (15) using a cut-point of 11.5 mm found 23% prevalence of LSS in 31 asymptomatic individuals. Other studies show different results, perhaps, due to different cutoff points. Most do not differentiate developmental stenosis from the degenerative type lumping them together. For example, Boden and colleagues (18) found DLSS in 1% of individuals younger than 60, and 21% in individuals over 60 years old in an MRI study of 67 asymptomatic individuals. Wiesel and colleagues (19) reported 50% of CT scans were abnormal among 52 asymptomatic individuals over 40 years of age. Leonid Kalichman (20) found a prevalence of absolute stenosis cut-off point 10 mm) to be 6.0% in asymptomatic individuals and 18.9% in individuals with LBP. Jarvik and colleagues (21) also found that severe LSS is less common in individuals without LBP and is likely to be diagnostically and clinically relevant.

Our study shows no difference in prevalence of radiographic LSS between men and women. This is consistent with findings in other studies (20,22). Jansson and colleagues (22) in a study among 11,283 cases also found no statistically significant differences between sexes. We can, therefore, conclude that there is no significant sex difference in the prevalence of LSS.

In this study, 57% of the LBP group had the vertebral body AP diameter smaller than 30mm at LV5

level compared to 48% of the control group. At LV4 44% had smaller bodies compared to 28% in the control group. We therefore, conclude that LSS is caused by failure of development of distal lumbar vertebrae in general while there appears to be a lag in growth of LV5 with failure to catch-up.

There are several drawbacks to this study; the first is using a single parameter (AP diameter) to measure the lumbar spinal canal. The second is that of limiting measurements to the distal lumbar canal. There are several limitations of the present study. First, is the use of two different scanning modalities; CT images for the control group and MRI images for the study group; assumedly introducing observer error? However, both modalities are considered reasonable alternative methods of evaluation of lumbar stenosis. Secondly, is the use of the antero-posterior diameter of the spinal canal alone, which may lead to underestimation of the prevalence of spinal stenosis, for example, in patients with trefoil shape of the spinal canal (23). Use of both the AP and lateral (transverse) diameters has been shown to be more accurate (20).

CONCLUSION

This study shows that a significant number of patients with chronic low back pain have a narrow spinal canal. The prevalence of absolute congenital lumbar spinal stenosis in this study is 7% (cut-off point of 10 mm) and relative stenosis of 26% at (cut-off point of 12 mm). There is a large schism in these findings with those of Leonid Kalichman (20) where they found prevalence rates of 4.71% and 2.62% for relative and absolute stenosis, respectively. The very high prevalence of LSS in this population may explain the high prevalence of neurological symptoms associated with chronic low back pain. It also poses a possibility of an aetiopathological process in this population that results in small lumbar spinal canals. This is the subject of a larger on-going study.

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